EDITORIAL

What is a Dream Generator?

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The lucid report by Wittman et al. in this issue (1) has examined the electroencephalographic (EEG) power spectra of non-REM (NREM) slow wave (SWS) and stage 2 sleep periods immediately preceding instrumental awakenings at which dream reports were solicited. Using a frequency-based index of cortical activation (less power below 8 Hz and more power above 12 Hz; see reference 2), these authors compared EEG activation from half-minutes preceding awakenings yielding dream reports to that preceding awakenings without such reports. In addition, for those awakenings yielding dream reports, they correlated the length (word count) of these reports with spectral power in the different frequency bands during the preceding half minute of sleep.

NREM epochs preceding awakenings yielding dream reports did not prove to be more activated than epochs prior to no recall using their operational criterion. However, correlations between report word count and spectral power in the different frequency bands were inversely significant for delta (0.75-4.5 Hz) power in one of two frontal and one of two central derivations. Sigma 2 (12.25-13.75 Hz) band power was positively correlated with word count in both posterior derivations, and beta 1 (14.0-22.0 Hz) and beta 2 (22.25-30.0 Hz) power were each positively correlated with word count in one of two posterior derivations. When, however, correlations were adjusted for time since lights out, only sigma 2 and beta 1 power remained positively correlated with report length (at one posterior derivation apiece). The authors conclude that while their data did not support an association of recall with cortical activation, spectral power correlations with word count did support a relationship between report length and cortical activation (i.e., longer reports were associated with more power in faster frequency bands, shorter reports were associated with more power in slower frequency bands).

Wittman et al. (1) interpret their correlation of report length with cortical activation as supportive of a one-generator versus a two-generator model of dreaming. One-dream-generator hypotheses assert that NREM and REM sleep dreaming differs "only quantitatively, not qualitatively" (e.g., 3-5) while two-generator hypotheses (e.g., 6-8) suggest that different brain mechanisms exist for dreaming in the two different sleep states (see reviews in 6, 9-11). Wittmann et al. bolster their argument of a one-generator mechanism with observations that: (i) NREM dreaming showed a high (60%) recall rate; (ii) dream recall or "a sense of having dreamt" followed most stage 2 awakenings; and (iii) sensory or bizarre features were present in most reports.

This carefully performed and clearly written study is an intriguing addition to the literature on the electrophysiological correlates of dreaming. However, interpreting findings such as these in regard to dream generator models encounters both logical and technological problems.

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Few and highly variable reports on dream length

It is important first to compare the quantitative aspects of REM and NREM dreams which are to be correlated with EEG features. (For the sake of clarity, and so as not to imply precise definitions where they do not exist, the term "dream" instead of "mentation" will be used throughout and no attempt will be made to differentiate dream production from dream recall.) In a recent review (6), only 3 of 24 studies reviewed (12-14) reported the actual mean word length of REM and NREM reports. The means of these mean word counts were 219 words for REM and 86 for NREM. Unfortunately, in other studies comparing REM and NREM dream length, comparisons were made between the number of sentences (15), a metric inherently imprecise with regard to word count. An even less reproducible metric, "temporal units," was also used to compare REM and NREM dreams in other studies (e.g., 5). Report of only this metric precludes accurate inter-study comparisons but its use has been perpetuated in later reports advocating one-generator theories (e.g., 16).

It is notable that Wittmann et al.'s (1) mean report length was only 18.2 words with a standard deviation of 14.2 and a maximum of only 57. When quantifying dream quantity with word counts, it is preferable to report the median as well as the mean because outlying high and low word counts are prevalent in both instrumental awakening and spontaneous dream reports (17). Nonetheless, it may be safely assumed that many of Wittmann et al.'s dreams must have been shorter than even their very low mean. Given the NREM mean from the above three studies, these reports may have been exceptionally short even for NREM dreams. They do, however, resemble word counts from Antrobus' 1983 study (3), the study most often cited as evidence for one-generator theories, in which median word count for NREM was only 7 words (see 11)! Notably, in the case of REM dreams, Germain et al. (18) classified fewer than 25 words (i.e., exceeding Wittmann et al.'s mean report length) as "no recall."

One- and two-generator theories of dreaming

Following the discovery of REM sleep by Aserinsky and Kleitman (19), the high probability of obtaining a dream report following REM awakenings led to early speculation that REM was the exclusive physiological substrate of dreaming (20). Soon afterward, however, sleep laboratory awakening studies revealed substantial recall of mentation from NREM sleep (21). Compared with NREM sleep, REM sleep is associated with greater dream report frequency, length, and bizarreness, as well as greater motoric and emotional intensity (reviewed in 6). Nonetheless, there is substantial recall of mentation from NREM sleep. In a review of 29 studies an estimated NREM recall rate of 42.5% has been contrasted with a REM recall rate of 81.8 % (9-10).

The one-generator versus two-generator controversy grew out of this lack of sleep stage specificity for obtaining dream reports. Essentially, one-generator hypotheses suggest that NREM dreams are shorter and less frequent than REM dreams simply because the cortex is less activated (e.g., 22-23). One generator theory rests largely on the observation that when word count is statistically partialled out, REM and NREM dreams show a similar frequency of dream features such as visual imagery and bizarreness (3,5).

Arguments that cortical activation cannot be the only determinant of dream quality ("two-generator" models in references 9-10) assert that factors such as the prevailing neuromodulatory milieu also contribute to dream quality as, for example, in Hobson et al.'s AIM model (6, 11). To summarize these "two-generator" arguments briefly, it is by no means clear that REM and NREM mentation are qualitatively the same for the following reasons: (i) Residual differences in dream features remain even after length is

controlled (6). (ii) Reports with more dream features will require more words to describe them thereby confounding dream length with dream complexity, bizarreness and intensity (24). (iii) There exist an abundance of cognitive differences between REM and NREM sleep that are unrelated to dreaming (10-11). Adding to these arguments is the fact that, in biological systems, more of an input (e.g., ascending reticular activation) does not necessarily produce more of the same output (e.g., a certain quality of dreams) as explained below.

Neilsen's theory of covert REM

Tore A. Nielsen's covert REM theory (10-11) provides an explanation for NREM dreaming which does not invoke simply a lower engagement of a universal mentation process in less activated brain states as postulated by one-generator theories (see 25). Nielsen defines covert REM as "any episode of NREM sleep for which some REM sleep processes are present, but for which REM sleep cannot be scored with standard criteria" (reference 10, p. 861). Although covert REM theory bears some similarity to the tonic-phasic model of sleep mentation (26-28), it is more comprehensive suggesting that REM-like brain processes may arise in many conditions not just those underlying phasic EEG, EOG or EMG events (see reference 28 for a comprehensive review of classical phasic sleep events). For example heart-rate variability in NREM increases with increasing temporal proximity to REM and, by increasing REM pressure using REM deprivation, the NREM sleep prior to REM can be made to exhibit yet more of an increase in heart-rate variability as well as more REM-anticipatory muscle atonia (29).

Possible conditions leading to covert REM and their accompanying physiological signs are reviewed extensively by Nielsen (9-10) as well as in Hobson et al. (6, 11). In addition, evidence of covert REM is provided in recent reports by Conduit and colleagues (30-32). This group first showed that stimuli applied below waking threshold elevate the frequency of reports of visual imagery during stage 2 NREM sleep to REM-like levels (30). Next, Conduit et al. (31) showed intercorrelation of eyelid movements (ELMS) with other phasic muscle movements and suggest both are facilitated by a common endogenous process such as the human equivalent of the feline ponto-geniculo-occipital (PGO) wave or a generalized CNS alerting mechanism. Most recently, this group has shown that stage 2 NREM awakenings preceded by ELMS yield higher frequency of visual imagery reports than awakenings not preceded by ELMS and that the amount of aroused EEG time (alpha or faster oscillations) immediately preceding ELMS awakenings was greater than aroused EEG time preceding no-ELMS awakenings (32). Interestingly, in REM, applied stimuli did not elevate imagery reports (30) and ELMS awakenings and their preceding EEG arousal time did not correlate with enhanced imagery reports (32). These authors suggest that phasically increased brain arousal associated with ELMS may enhance recollection of stage 2 mentation but that, in REM, arousal is tonically at levels sufficient for imagery recall (32).

EEG spectral correlates of dreaming

EEG spectral analysis of sleep preceding NREM awakenings, such as performed by Wittmann et al., asks whether levels of operationally defined EEG arousal or power in arousal-related EEG frequencies can predict occurrence and quantity of NREM dream recall. Since a higher frequency and lower-amplitude EEG is characteristic of REM compared to NREM, such spectral analyses of NREM sleep may be equivalent to a search for covert REM.

Not all NREM sleep studies have found the spectral band power correlations with dreaming reported by Wittmann et al. (1). These authors cite the study of Williamson et al. (33) as an example of a positive

association between cortical activation and NREM dream recall. However, Williamson et al. did not find differences in spectral power, within the delta-theta, alpha and beta bands they examined, between Stage 2 NREM awakenings with dream recall and those without recall (33). Instead, they reported a non-significant tendency for power in delta-theta and beta bands to decrease with increased dream recall. (Note, however that a generalized decrease in power has sometimes been interpreted to represent activation, see 34-35). Despite no spectral power differences, Williamson et al. did find that, within their beta band (13-26 Hz), the mean beta frequency was higher with more dream-like recall (33). Similarly, Morel et al. (36) failed to find evidence that greater activation in Stage 2 sleep preceding awakenings was associated with dream recall (although there were post-awakening spectral differences between recall and no-recall conditions).

In some studies of REM sleep, awakenings with dream recall have shown lower power across a broad range of frequencies compared to awakenings without recall (34-35). Authors of these two reports advocate the Functional State Shift hypothesis (37) which predicts that greater activation and a more wake-like physiology in sleep leads to more dream recall (34-35,37). Similarly, Germain et al. (18) found that REM dream recall was associated with greater power in faster frequencies (alpha, beta 1 and 2) but found no association of recall with power in lower frequency bands. In contrast, Wollman and Antrobus (2) failed to find any association between total word count in REM and power in delta, theta, alpha, sigma or beta frequencies although they did show an inverse correlation between visual elements and delta power.

Therefore for both NREM and REM awakenings, the association of increased high frequency and/or decreased low frequency spectral power with dream recall and/or report length remains somewhat equivocal. Nevertheless, Takeuchi et al. (8) notes that the general weight of evidence suggests that the faster EEG frequencies (beta and gamma) are associated with dream recall. The association of REM (but not NREM) sleep with gamma frequency (30-80 Hz) oscillations using EEG (38) and MEG (39) is especially intriguing. Gamma range oscillations are associated with attention and cognitive effort in waking (40), and have been hypothesized to also be associated with the temporal binding of dream imagery (41). Takeuchi's sleep interruption experiments have shown that sleep onset REM and NREM dreams have markedly different EEG correlates with sleep onset REM dreams associated with REM activation processes and sleep onset NREM dreams associated wake-related arousal processes (7-8). Interestingly, however, spectral correlates of hypnogogic images involve increases in delta power and decreases in power of all other bands (42) suggesting that decreased EEG arousal favors such imagery earlier in sleep onset.

EEG activation, NREM dreaming and covert REM

One possible explanation for inconsistent findings on spectral correlates of NREM dreaming is that covert REM-like processes are not always present in this stage of sleep (10). Therefore, in the studies of Wittmann et al. (1), Williamson et al. (33) and Morel et al. (36) in which time into NREM is used as an awakening criterion, the investigators may or may not have conducted awakenings when such dream-producing (or dream-recall-enhancing) processes were present. In contrast, Conduit et al. (32) conducted awakenings based upon ELMs, a REM-related sign (43) and, hence, achieved elevated reports of imagery. Notably, Conduit et al. (32) found less EEG arousal preceding no-ELM compared to ELM awakenings during stage 2 sleep (albeit using a measure less precise than spectral analysis). In contrast, during REM sleep, both the imagery-ELM and ELM-EEG arousal associations were absent (32) nor

could stimuli enhance imagery (30).

Covert REM theory explains the above observations as follows: (i) In stage 2, ELMs indicate presence of transient arousal and, perhaps, other phasic events (e.g., PGO waves) and hence correlate with imagery recall (e.g., 30, 32). (ii) Stage 2 awakenings not based on such signs may miss such transient arousal (and/or REM-like process) and hence not demonstrate an association of dream recall with EEG features (1, 33, 36). However, if dreaming is present before awakening, it's intensity (33) or length (1) may be associated with EEG features. (iii) REM dreaming shows less association with spectral power (2) or ELMs (30, 32) because the more activated REM conditions (indexed by a higher frequency, lower amplitude EEG signal) are already optimal for dream production or recall (32).

The limitations of dream generator theories

In human electro- and magnetoencephalography, "generator" may refer to: (i) a discrete brain structure pinpointed by intracranial EEG (e.g., 44); (ii) a discrete brain structure inferred by dipole tracing techniques (e.g., 45); (iii) approximate anatomic coordinates of dipole sources of particular wave form (e.g., 46); (iv) distributed structures known to generate a characteristic wave form such as the thalamocortical generator of spindles (e.g., 47); (v) a poorly defined network of forebrain structures which never-the-less generate a distinct wave form (e.g., 48); or any other operationally defined source of an electrical or magnetic signal.

None of these definitions, however, capture what is implied by "dream generator" in the context of the one versus two-generator debate. Rather, “dream generator” refers to the totality of neural events producing the subjective experience of dreaming and the physiological signs that are concurrently measured. It is reasonable to assume that, despite blockade of exteroceptive inputs, dream experience, like waking consciousness, is the product of interacting, distributed networks subserving different cognitive processes such as spatial sense, memory, emotion, language, object recognition, and executive function as described by Mesulam (49-50). In generating waking cognition, the activity of different networks are dependent upon certain key nodes of converging neural activity in a mode of organization described by Mesulam as “selectively distributed processing” (49-50). Again, it is parsimonious to assume that these same nodes are key to cognitive processes and fictive experiences in dreaming. Activity in these major nodes (e.g., Wernicke’s and Broca's areas for language networks) is typically clearly visualized by functional neuroimaging of tasks requiring their corresponding cognitive skills (e.g., language comprehension and production respectively in the above example, see 50-51).

In both one- and two-generator dream models, the operation of these cognitive networks, in sleep, are assumed to be more activated during dreaming than when dreaming is not occurring, presumably due to increased activity in at least a subset of the same ascending arousal systems responsible for network activation in waking. In addition, both hypotheses acknowledge that these networks are largely deprived of external and somatic sensory input during dreaming. pon this basis, however, two-generator theories add that physiological conditions differ during dreaming in different sleep states and that this, in some way, qualitatively alters the subjective dreaming experienced in different states (6).

The most obvious basis for such physiological differences are differences in the neuromodulatory influences on forebrain structures prevailing during different sleep states as elaborated in the AIM model (6). The existence of such neuromodulatory differences between mammalian sleep states has been repeatedly demonstrated in animal models

and, indeed, also in some pioneering human microdialysis studies (reviewed in 6). Following sleep onset and subsequent deepening of NREM sleep, there is a corresponding decline in activity of arousal-related neurons in ascending aminergic (noradrenergic, serotonergic histaminergic) and cholinergic influences that widely influence forebrain regions via projections from brainstem, hypothalamic and basal forebrain nuclei (see 52 for a review). In REM, however, aminergic influences reach their nadir while brainstem cholinergic neurons resume activity re-exciting the reticular core and diencephalon which, in turn, produce the re-activation of forebrain regions observed in REM (6,52). Influences on these very neuromodulatory systems constitute the bases for activity of most psychoactive drugs (53) and, hence, sleep-stage related changes in these systems might also be expected to alter dream experience in a state-dependent manner (53 see also 54). Indeed pharmaceutical manipulations of these systems result in marked dream changes (55-56).

However, even if such differential neuromodulatory modes of forebrain activation have no differential effects on dream experience (as one-generator dream models suggest), increasing levels of activity in neuronal systems do not necessitate a homogeneous, monotonic increase in the quality of their output. Indeed, qualitative changes in neuronal systems based upon activation thresholds are often postulated. As one example, different synaptic levels of a neurotransmitter may activate entirely different receptor systems (57). Similarly, there exist thresholds of thalamocortical activation by ascending arousal systems below which the low frequency thalamocortical oscillations (i.e., spindle, delta, slow oscillation) can emerge (58-59). These oscillations are again blocked when ascending activation subsequently exceeds such thresholds with the onset of REM sleep (58-59). Therefore, even “activation-only” could result in both a physiologically and phenomenologically different dream generator in the more activated REM state compared to less activated NREM. Recruitment of entire forebrain networks or groups of networks at certain activation thresholds might produce qualitatively different dream experiences in different, sleep-stage dependent activation states (i.e., a two-generator model). It seems, however, equally likely that sleep-stage dependent neuromodulatory changes would also influence subjective dream experience for all the reasons elaborated above.

Dichotomization of dream theories into one and two "generators" (like the dichotomization of sleep into REM and NREM) threatens a gross simplification of CNS reality as well as an inappropriate reification of what currently are only heuristic concepts. Quantitative and qualitative variations in macroscopic regional brain activation patterns (and their corresponding phenomenological output) are manifold whether "activation-only" or "activation-plus-neuromodulation" globally influences dream quality. In the following discussion, only changing activation levels of different network configurations need be assumed. However, the neuromodulatory effects undoubtedly present would be expected to further multiply the number of potential “qualitatively different” brain and dreaming states. And in either case, scalp EEG may not be sensitive or reliable enough to define such differences.

If the dream generators are interpreted as a series of events in discrete brain structures (e.g., Mesulam’s key nodes in selectively distributed processes, 49-50), reliance upon scalp EEG measurements for the detection and identification of these loci is a weak approach for the following reason. To observe brain events corresponding to an endogenously initiated process, for example a steady potential shift ERP like the readiness potential, measured EEG activity must be both time locked to a fixed event and summed over numerous trials in order to cancel the more powerful, random background activity signal (60). Assuming

that dream elements (e.g., visual imagery) reflect activation of particular sensory or multimodal association areas localized in time and space (e.g., downstream ventral and dorsal stream visual association areas), then neither of these measurement conditions are fulfilled in EEG dream studies except, perhaps, if EEG activity is locked to a peripheral sign such as ELMS (32). Only recently, are physiological measures with both good temporal and good spatial resolution (such as MEG tomography) being applied to events relevant to dreaming (e.g., REM saccades) occurring in deep subcortical structures (61).

If the dream generators are interpreted as sets of interacting, selectively distributed neural networks (49-50, see above), then the presence of multiple networks with both separate and overlapping nodes of activity must considered when interpreting electrophysiological or neuroimaging data. The most striking difference between brain activation patterns in REM versus NREM sleep lies in the much greater activity of limbic networks during REM (62-66). These are deep structures whose activity is difficult to separately identify at the level of scalp EEG. Although characteristic activity in such medial areas (e.g., midline theta) can be reliably reproduced under carefully controlled cognitive testing conditions in waking (67), the dreaming state, occurring as it does during sleep, precludes such experimental controls.

Lastly, if a dream generator is interpreted simply as all brain activity leading to dream experience and its accompanying physiological signs, then the "generator" becomes the integrated activity of all the brain structures, networks and neurochemistry concurrent with the dream experience. When activity of all central neural systems is thus summed, the generator becomes the brain itself which, of course, is unitary and a "single generator," but one with a near infinite range of configurations. As such, any "singleness" of this dream generator is meaningless just as the waking brain-mind is multi-dimensional and can assume any number of psychological and physiological configurations.

A thought experiment on dream generators

Imagine two REM dream experiences one involving the face of a loved one with whom you are conversing with intense emotion and the other where you calmly inspect a detailed, vivid landscape filled with objects and a varied topography. In both cases, fast EEG frequencies (beta, gamma) are observed to be elevated, presumably by arousal associated with cognitive-perceptual processing. Assume, as well, that cognitive processes in dreaming are subserved by similar brain networks as in waking (a logical extension of imagery and hallucination studies, see reference 6, as well as of one-generator dream theories). In the first dream, high frequency EEG activity reflects activation of inferior temporal fusiform (face), superior temporal (language) and midline limbic structures (emotion), whereas in the other dream, it reflects activity of dorsal stream (parietal) and ventral stream (temporal) visual association areas. Activation of these areas in dreaming is not inconceivable given the key importance of inferior parietal areas to dreaming (68) and the activation of both visual association and midline limbic areas in REM (63). Since EEG (unlike MEG) signals are "smeared" by the low conductivity of the skull (69), it is most unlikely that any measurable surface EEG activity would reliably differentiate these two dream experiences. But with the exception of ventral stream areas, the networks activated by these two dreams differ markedly and, if such a thing were possible, these two experiences would in all likelihood be accompanied by far different fMRI activation patterns. Now, do these two psychophysiological events reflect one or two generators? The question is meaningless because the possibilities for different generators are infinite!

Cognitive neuroscientific study of dream generators

It may be that dream researchers will need much patience in developing precise, empirical and brain-based models of dream generation. Unlike identification of global behavioral states (e.g., REM and NREM sleep), scalp EEG wave forms and power spectra may simply be inadequate to discriminate between different global mental processes (e.g., REM and NREM dreaming). By analogy with the cognitive neuroscience of waking, the remarkable findings now emerging from elaborate, event-related fMRI protocols using powerful magnets were, in the early to mid-nineties, inconceivable using contemporary neuroimaging technologies (PET, SPECT, early fMRI). Since dreaming (and/or the ability to recall dreaming) is not present at all points during sleep, a degree of temporal accuracy is needed for the interpretation of spatial functional images. In the study of cognitive processes in waking humans, multi-modal imaging approaches (e.g., EEG/MEG anatomically constrained by structural or functional neuroimaging) are now the gold standard for obtaining good resolution in both temporal and spatial domains (70-71). Since sleeping humans are not conscious, an external physiological sign is needed to mark times when dreaming is occurring. Providing such temporal markers for applying more advanced imaging technologies may prove to be the essential utility of knowing the EEG correlates of dreaming, an important knowledge base built upon studies such as that of Wittmann et al.

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